PROGRAMME BOOK

NEUR 2014 SCIENCE

25TH ANNUAL SCIENTIFIC MEETING OF MALAYSIAN SOCIETY OF NEUROSCIENCES

20th - 22nd June 2014 • Berjaya Times Square Hotel, Kuala Lumpur

Organised by

MALAYSIAN SOCIETY OF NEUROSCIENCES
persatuan neurosains malaysia

Once a day
for a full day of function

SIFROL® ER

C: Pramipexole diHCl monohydrate **I:** Tab & ER Tab Treatment of signs & symptoms of advanced idiopathic Parkinson's disease as monotherapy or in combination w/ l-dopa. ER tab Dose escalation: 0.375 mg on wk 1, 0.75 mg on wk 2, 1.5 mg on wk 3. Increase by 0.75 mg at wkly intervals if needed up to a max of 4.5 mg/day. *Patient on l-dopa* Reduce dose. *Renal impairment: CrCl 30-50 mL/min* Initially 0.375 mg every other day. May be increased by 0.375 mg at wkly intervals to max 2.25 mg/day. **A**:d: May also be taken w/ meals to minimise GI upset. ER tab: Swallow whole, do not crush/chew. **SP:** Renal impairment, severe CV disease. Behavioural changes may occur (reflecting symptoms of impulse control disorders & compulsive behaviours eg binge eating, compulsive shopping, hypersexuality&pathological gambling). Dose reduction/taper discontinuation should be considered. Pathologic changes. Avoid abrupt w/drawal. Pregnancy & lactation. May impair ability to drive or operate machinery. **AR:** Nausea, constipation, somnolence, hallucinations, confusion, dizziness, dyskinesias, hypersexuality, other abnormal behaviour, amnesia, compulsive shopping, restlessness, visual impairment including diplopia, blurred vision or reduced visual acuity, vomiting, wt decrease including decreased appetite, wt increase, disorders of libido, headache, pruritus, rash, syncope. **DI:** * Cimetidine, amantadine & other sedating drugs or alcohol.

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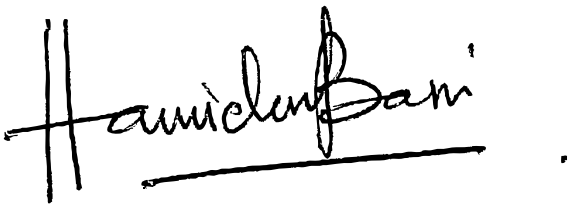
MESSAGE FROM THE ORGANISING CHAIRMAN

Welcome to the MSN 2014 annual scientific meeting. This year our meeting is in Kuala Lumpur and we will be co-organising the Mohandas Memorial Lecture with the Neurosurgical Association of Malaysia. We also have prepared various symposium topics in movement disorders, stroke, epilepsy and neuromuscular diseases. We also have case discussion and neurology examination teach-in sessions for postgraduates. There is a session on frontiers of neuroscience research which will showcase the many works done by our colleagues especially in basic sciences. I am sure that this meeting will be beneficial to all members of the society. I wish everyone a good and successful meeting.



Hamidon Basri
Organising Chairperson
25th Annual Scientific Meeting of Malaysian Society of Neurosciences

ORGANISING COMMITTEE

ORGANISING CHAIRPERSON Hamidon Basri

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COMMITTEE MEMBERS Irene Looi
Lim Kheng Seang
Michael Ling King Hwa
Mohd Feizel Alsiddiq Mohd Fakharuddin
Ooi Phaik Yee
Santhi Datuk Puvanarajah

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- Alex Khoo Peng Chuan
- Azlina Ahmad Annuar
- Cheah Pike See
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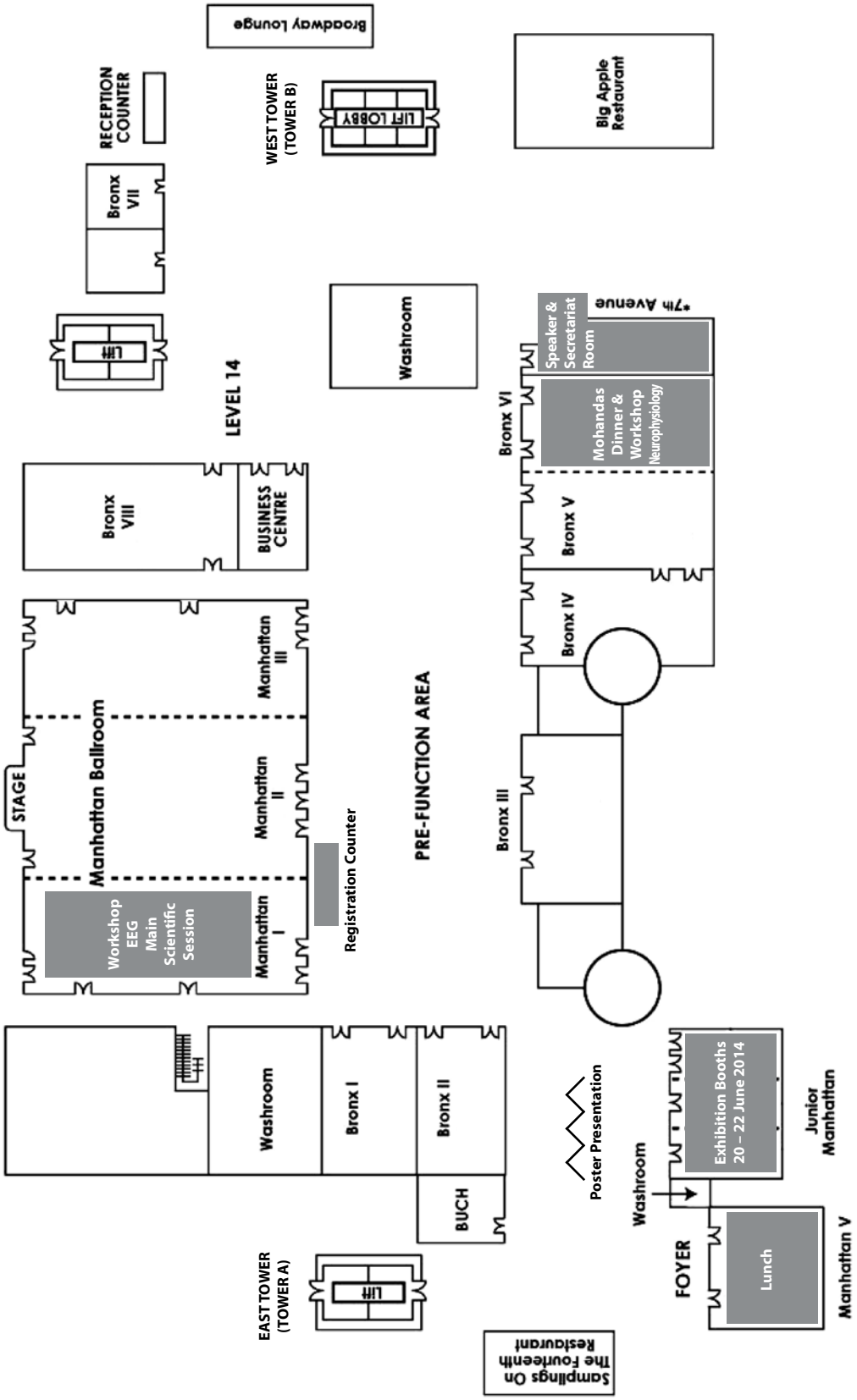
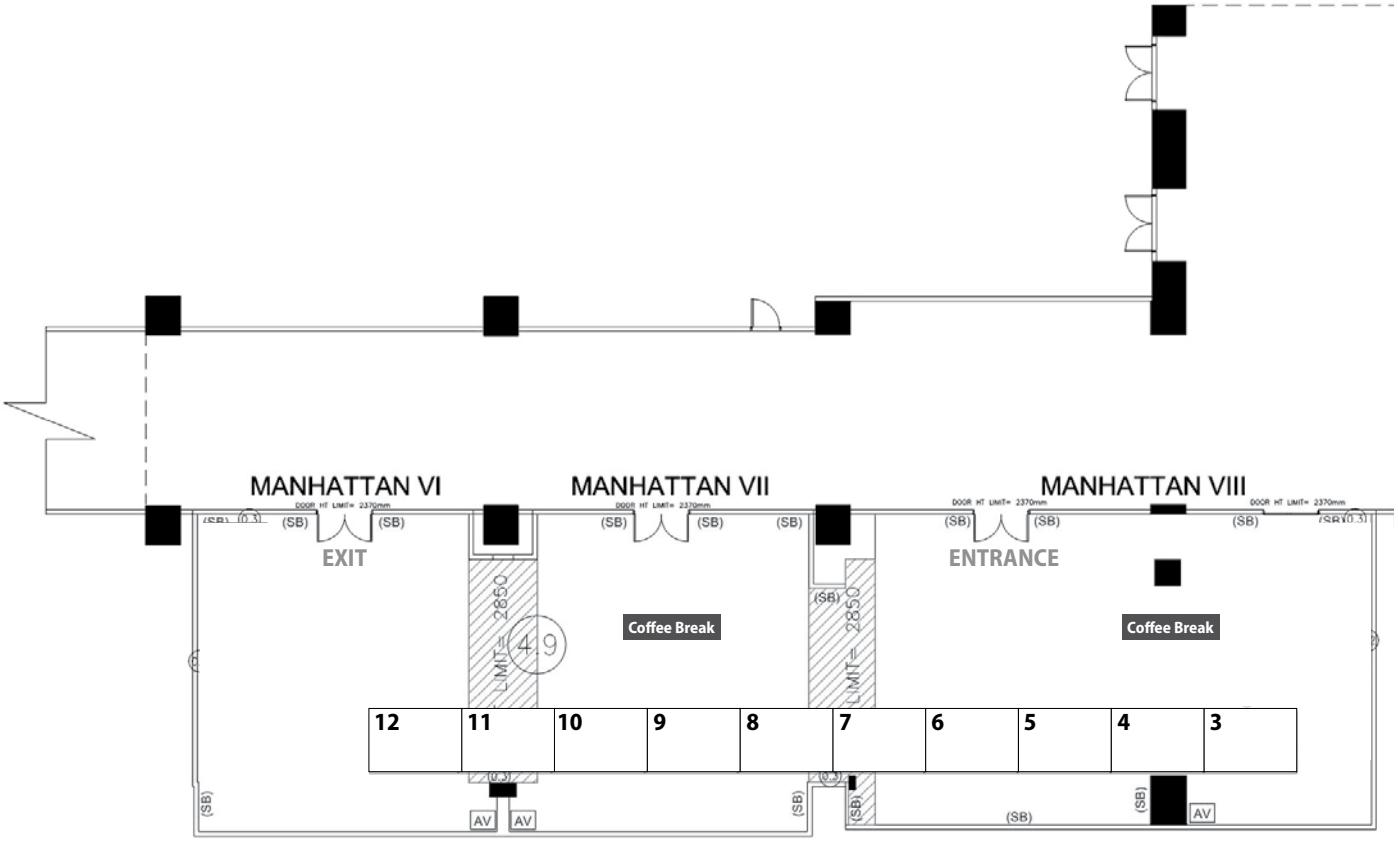
- Raymond Tan
- Santhi Datuk Puvanarajah
- Siva Seeta Ramaiah
- Tajul Arifin
- Tan Chong Tin
- Tan Hui Jan
- Teh Chee Ming

VENUE LAYOUT PLAN

Function Room, Level 14, Berjaya Times Square Hotel



EXHIBITION FLOORPLAN



Company	Booth No.
Pfizer (Malaysia) Sdn Bhd	3
Hovid Pharmacy Sdn Bhd	4
GlaxoSmithKline Pharmaceutical Sdn Bhd	5
Novartis Corporation (M) Sdn. Bhd.	6
Eisai (Malaysia) Sdn Bhd	7
DanMedik Sdn Bhd	8
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CONFERENCE INFORMATION

Registration Counter
Registration Counter is located at **Foyer of Manhattan I, Level 14, Berjaya Times Square Hotel.**

Opening hours of the Registration Counter

20th June 2014 Friday	1200 hr - 1730 hr
21st June 2014 Saturday	0730 hr - 1730 hr
22nd June 2014 Sunday	0730 hr - 1230 hr

Trade Exhibition
Trade Exhibition is located at **Junior Manhattan, Level 14, Berjaya Times Square Hotel** and the opening hours are as follows:

20th June 2014 Friday	1430 hr - 1730 hr
21st June 2014 Saturday	0730 hr - 1730 hr
22nd June 2014 Sunday	0730 hr - 1230 hr

Free Paper (Poster) Presentation
Free Paper (Poster) presentation is located at **Foyer of Manhattan I, Level 14, Berjaya Times Square Hotel** and and the opening hours are as follows:

20th June 2014 Friday	1430 hr - 1730 hr
21st June 2014 Saturday	0730 hr - 1730 hr
22nd June 2014 Sunday	0730 hr - 1230 hr

Official Language
The official language of the Conference is English.

Certificate of Attendance
Certificate of Attendance will be given to all registered delegates at the Registration Counter.

Name Badges
Registered delegates are to wear their name badges at all times during the Conference for identification and security purposes. Admission to all Conference sessions and official functions is based on name badges.

Cellular Phone
As a courtesy to all delegates and speakers, cellular phones, pagers and others electronic devices must be operated in silent/vibrated mode throughout the Conference sessions. No telephone conversations are permitted in the session rooms.

Lunch
• All the Lunch will be served at **Manhattan V, Level 14, Berjaya Times Square Hotel**.
• Please present your lunch voucher to the staff on duty to enter the Lunch area.

Coffee Break
Morning and evening coffee break will be served at the **Trade Exhibition** located at **Junior Manhattan, Level 14, Berjaya Times Square Hotel.**

Liability
The Organising Committee will not assume any responsibility for accidents, losses or damages, as well as for delays or modifications in the programme.

PRE-CONGRESS WORKSHOP

20th June 2014, Friday

	PROGRAMME	
1430-1500	*Venue: Bronx VI, Level 14, Berjaya Times Square Hotel* **Workshop- Neurophysiology 1** ***Chairperson: Dr. Ong Beng Hooi*** 1. Electrodiagnosis of Polyneuropathies *Dr. Rabani Remli*	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Workshop- EEG 1** ***Chairperson: Prof. Dato' Tan Chong Tin*** 1. Look Before You Leap- The Pearls in EEG Reading *Prof. Dato' Dr. Raymond Azman Ali*
1500-1530	2. Diagnostic Approach to Focal Peripheral Neuropathies *Dr. Hiew Fu Liong*	2. When to Order EEG? *Dr. Raymond Tan*
1530-1600	*Venue: Junior Manhattan, Level 14, Berjaya Times Square Hotel* **Coffee/ Tea Break**	
1600-1630	*Venue: Bronx VI, Level 14, Berjaya Times Square Hotel* **Workshop- Neurophysiology 2** ***Chairperson: Dr. Rabani Remli*** 3. Electrodiagnosis of Myopathies and Neuromuscular Junction Disorders *Dr. Ong Beng Hooi*	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Workshop- EEG 2** ***Chairperson: Dr. Raymond Tan*** 3. Normal EEG Patterns in Adult *Assoc. Prof. Dr. Tan Hui Jan*
1630-1700	4. Case Demonstration	4. Normal and Important EEG Patterns in Children *Dr. Ahmad Rithauddin*
1700-1730		5. Ictal EEG *Prof. Dato' Dr. Tan Chong Tin*
1900-2015	*Venue: Bronx VI, Level 14, Berjaya Times Square Hotel* **Mohandas' Memorial Lecture (For Invited Guests Only)** Endoscopic Neurosurgery of the Intracranial Potential Spaces *Dr. Azmi Alias*	
2015-2200	*Venue: Bronx VI, Level 14, Berjaya Times Square Hotel* **Dinner (For Invited Guests only)**	

SCIENTIFIC PROGRAMME

Day 1 | 21st June 2014, Saturday

	PROGRAMME
0730-0830	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Teach-In Session: Postgraduate Neurology Examination Tips- Cranial Nerves** *Prof. Dato' Dr. Raymond Azman Ali*
0830-0900	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Plenary Lecture Sponsored by Hovid Pharmacy** Tocotrienols and Neuroprotection *Dr. Wong Jia Woei*
0900-0930	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Epilepsy Symposium** ***Chairperson: Dr. Santhi Datuk Puvanarajah*** 1. 3F- Differential Diagnosis of Blackouts *Prof. Dato' Dr. Raymond Azman Ali*
0930-1000	2. Use of Newer AEDs and Treatment Modalities in Malaysia *Assoc. Prof. Dr. Lim Kheng Seang*
1000-1030	3. Epilepsy in Women *Datuk Dr. Raihanah Khalid*
1030-1100	*Venue: Junior Manhattan, Level 14, Berjaya Times Square Hotel* **Coffee/ Tea Break and Healthcare Industry Exhibition**
1100-1130	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Stroke Symposium** ***Chairperson: Dato' Dr. Hj. Md. Hanip bin Rafia*** 1. Paediatric Stroke : Challenges in Diagnosis and Treatment *Dr. Teh Chee Ming*
1130-1200	2. Acute Stroke Management *Dr. Siva Seeta Ramaiah*
1200-1230	3. Secondary Prevention of Stroke: Anti-platelet Therapy and its Rational Use *Prof. KS Tan*
1230-1400	*Venue: Manhattan V, Level 14, Berjaya Times Square Hotel* **Lunch**
1400-1430	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Movement Disorders Symposium** ***Chairperson: Dr. Irene Looi*** 1. Updates on the Management of Parkinson's Disease *Dr. Ooi Phaik Yee*
1430-1500	2. Approach to Movement Disorders in Children *Dr. Tajul Arifin*
1500-1530	3. Hyperkinetic Movement Disorders *Prof. Datin Dr. Norlinah Ibrahim*
1530-1600	*Venue: Junior Manhattan, Level 14, Berjaya Times Square Hotel* **Coffee/ Tea Break and Healthcare Industry Exhibition**
1600-1730	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Case Discussion** ***Panel: Prof. Dato' Dr. Raymond Azman Ali , Prof. Dato' Dr. Tan Chong Tin, Prof. KJ Goh*** *Cases from UM, HKL & UPM*
1730-1900	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **MSN AGM**
1900-2100	*Venue: Manhattan V, Level 14, Berjaya Times Square Hotel* **Dinner Symposium by Pfizer** NOACs for Stroke Prevention in Atrial Fibrillation - What's New? *Dr. Lawrence Wong*

SCIENTIFIC PROGRAMME

Day 2 | 22nd June 2014, Sunday

	PROGRAMME
0730-0830	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Teach-In Session:** **Postgraduate Neurology Examination Tips- General Examination** *Prof. Goh Khean Jin*
0830-0900	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Research Frontiers in Neuroscience** ***Chairperson: Dr. Michael Ling*** 1. Neuroscience Research Programme @ UPM *Dr. Cheah Pike See*
0900-0930	2. A Pushy Partnership-Endocannabinoid and Ketum *(Mitragynaspeciosa)* Misuse Potential *Dr. Muzaimi Mustapha*
0930-1000	3. From Mapping Tiny Neurons to Mapping Cognitive Processes *Dr. Azlina Ahmad Annuar*
1000-1030	*Venue: Junior Manhattan, Level 14, Berjaya Times Square Hotel* **Coffee/ Tea Break and Healthcare Industry Exhibition**
1100-1130	*Venue: Manhattan I, Level 14, Berjaya Times Square Hotel* **Neuromuscular Diseases Symposium** ***Chairperson: Assoc. Prof.Dr. Nortina Shahrizaila*** 1. Update on Muscle Disorders *Prof. Goh Khean Jin*
1130-1200	2. The Role of Immunotherapy in Neuromuscular Disorders *Dr. Santhi Datuk Puvanarajah*
1200-1230	3. Evaluation of a Floppy Child : From the Bedside to Workbench *Dr. Alex Khoo Peng Chuan*
1230	**Closing**

SPEAKER ABSTRACTS

Date: 20th June 2014
Time: 1430 - 1500
Pre-Congress Workshop - Neurophysiology 1
Venue: Bronx VI, Level 14, Berjaya Times Square Hotel

ELECTRODIAGNOSIS OF POLYNEUROPATHIES

Dr. Rabani Remli
Clinical Specialist Neurology and Lecturer at the UKM Medical Centre, Kuala Lumpur.

Electrodiagnostic testing follows from the history and is an extension of the neurologic examination. When clinical assessment implicates a peripheral neuropathy, the goal of electrodiagnostic testing is to more fully characterize the neuropathy in terms of the distribution (motor, sensory, symmetric, or asymmetric), extent of a neuropathy (symmetric, legs, or arms), and time course (very chronic or ongoing). Of greatest importance is that electrodiagnostic testing should help identify the type of underlying pathologic condition (primary axonal, primary demyelinating, or conduction block). Once this is accomplished, the differential diagnosis narrows and rational laboratory testing can be ordered or treatment trials initiated.

Date: 20th June 2014
Time: 1500 - 1530
Pre-Congress Workshop - Neurophysiology 1
Venue: Bronx VI, Level 14, Berjaya Times Square Hotel

DIAGNOSTIC APPROACH TO FOCAL PERIPHERAL NEUROPATHIES

Dr. Hiew Fu Liong
Hospital Kuala Lumpur

Each peripheral nerve, by virtue of its unique course is vulnerable to injuries from various causes at many points along their course. Frequently in our daily practice, the diagnosis is made, as always, by careful history and clinical assessment, including observation of the pattern of weakness, sensory loss and atrophy of the muscles supplied by a particular nerve distal to the lesion. Therefore, knowing the anatomy of these nerves is essential in diagnosing focal neuropathies. While some focal compression neuropathies come with straightforward cause e.g. Carpal Tunnel Syndrome, this clear distinction may be blurred in certain types of neurological diseases that, in early stages manifested as a localized pathology at the common sites of compression. Thus, ancillary procedures such as nerve conduction studies and needle electromyography may add to the accuracy of the diagnosis.

This overview will explore the general approach to the diagnosis of focal peripheral neuropathies, including the use of electrophysiology study to help localize and characterize a focal lesion in a proper clinical context.

SPEAKER ABSTRACTS

Date: 20th June 2014
Time: 1430 - 1500
Pre-Congress Workshop - EEG 1
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

LOOK BEFORE YOU LEAP: THE PEARLS IN EEG READING

Prof. Dato' Dr. Raymond Azman Ali
Senior Consultant Neurologist
Department of Medicine
Universiti Kebangsaan Malaysia Medical Centre

Normal EEG variants are "unusual" cerebral activities occurring in otherwise normal individuals. They may mimic epileptic discharges and therefore lead to misinterpretation of the record. Artifacts are physiological or pathological extracerebral activities or "noise" created by the EEG or nearby electrical equipment. The recognition of variants and artifacts is of paramount importance in order that the final EEG report is not an overinterpretation of the true cerebral activity.

Normal variants are uncommon before the age of 21 months and after adolescence. Between these two ages, "unusual" rhythms are common, and accepted as normal variants because they are generally not associated with abnormal clinical findings or increased propensity for future epilepsy. They are most commonly seen in sleep and are somewhat age-dependent, and include: rhythmic hypnagogic theta of drowsiness, extreme sleep spindles, anterior rhythmic theta activity, 14 and 6/sec positive spikes, psychomotor variant, 6/sec spike-wave complexes, benign epileptiform transients of sleep, and wicket spikes. Posterior rhythms that are considered to be normal variants include slow and fast alpha variants, posterior slow waves of youth, lambda waves, and positive occipital sharp transients of sleep. Low voltage records and slow alpha variants are not uncommon in adolescence.

Artifacts are most often caused by movement and muscle activity due to swallowing, teeth clenching, chest movements, blinking and eyelid flutter, and hand tremor. EEG recording in ICU is especially difficult because of artifacts caused by the ventilator, intravenous infusion pumps, ECG, etc. Artifacts may also be caused by pathological extracerebral activity like hemifacial spasm.

When reporting the EEG, it is strongly recommended to use phrases like "within normal limits (for age)" rather than use rigid conclusions about normalcy and abnormality. It is important to recognise what is normal and the normal variants, and what is of cerebral origin and what is extracerebral. Annotation by the EEG technologist is of utmost importance in this respect. Finally, always under-report the EEG, and thus, "look before you leap!

SPEAKER ABSTRACTS

Date: 20th June 2014
Time: 1600 - 1630
Pre-Congress Workshop - Neurophysiology 2
Venue: Bronx VI, Level 14, Berjaya Times Square Hotel

ELECTRODIAGNOSTIC (EDX) APPROACH TO MYOPATHY AND NEUROMUSCULAR JUNCTION DISORDERS

Ong Beng Hooi
Department of Medicine, Neurology unit, Hospital Sultanah Bahiyah, Alor Setar, Kedah, Malaysia.

Myopathies often pose a diagnostic challenge as a differential causes for weakness. Electrodiagnostic (EDX) is very useful in differentiating myopathy from neurogenic causes of weakness that may mimic myopathy. In this respect, EDX is always an extension of physical examination and clinical history. Here we look into the needle EMG examination for myopathy; the four main components include assessment of (1) insertional activity, (2) spontaneous activity, (3) MUAP morphology and (4) recruitment. Then we elaborate on the general EMG pattern group in helping to formulate the differential diagnosis.

EDX examination in patients with suspected NMJ disorders requires a good comprehension of physiology and pathophysiology of neuromuscular transmission. Our aim is to look into the basic practical knowledge of neuromuscular transmission as it relates to the EDX studies; the RNS and SFEMG and, elaborate on the technical aspect of these neurophysiological tests.

The main goal of this lecture is to provide clinical and technical understanding of EDX in of myopathies and NMJDs, which that can assist neurophysiologists and technicians in their daily neurophysiology work.

Date: 20th June 2014
Time: 1600 - 1630
Pre-Congress Workshop - EEG 2
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

THE NORMAL EEG PATTERNS IN ADULTS

Assoc. Prof. Dr. Tan Hui Jan
UKM

The electroencephalograph (EEG) is a useful tool in the management of epilepsy patients. The routine 10-20 recording is a standard procedure for analysis of the brain activity. The electrodes consist of Fp2, F4, C4, P4, O2,F8, T4, T6, Fpz, Fz, Cz, Pz, Oz, Fp1, F3, C3, P3, O1, F7, T3, T5. Fp stands for frontopolar, F for frontal, C for central, P for parietal, O for occipital and T for temporal. The EEG report is a formal and orderly process that describes the background activity, hyperventilation and intnermittnet photic stimulation, physiological sleep patterns and presence of variants or abnormalities. The background activity refers to the referential EEG corresponding to the patient's age and state of consciousness. The background activity can be observed in the posterior quadrants when eyes are closed. The alpha rhythm frequency ranges from 8-13 c/s, beta frequency over 13 c/s, theta frequency ranges from 4-7 c/s and delta frequency under 4 c/s. Hyperventilation and intermittent photic stimulation are activation procedures performed for inducing and recording physiological or pathological abnormalities. A sleep recording can be performed during the EEG. The stages of sleep include non-REM sleep (light sleep and deep sleep) and REM sleep. The writing of the EEG report is based on the analysis of the recording and arriving at the conclusion.

SPEAKER ABSTRACTS

Date: 20th June 2014
Time: 1630 - 1700
Pre-Congress Workshop - EEG 2
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

NORMAL AND IMPORTANT EEG PATTERNS IN CHILDREN

Dr. Ahmad Rithauddin
Paediatric Institute Hospital Kuala Lumpur

Paediatric EEG differs from Adult EEG because the electrical activity of the brain changes during growth and development. In addition, there are differences in terms of head size and thickness of skull, as well as the child's behaviour and ability to cooperate, all of which contribute to the specific EEG patterns observed in children. There is a much wider range of normality, as well as specific age related abnormalities seen in the paediatric population. This talk will discuss some of the important normal and abnormal patterns of paediatric EEG.

Date: 21st June 2014
Time: 0900 - 0930
Epilepsy Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

3 Fs – DIFFERENTIAL DIAGNOSIS OF BLACKOUTS

Prof. Dato Dr. Raymond Azman Ali
Senior Consultant Neurologist
Department of Medicine
UKM Medical Centre

Not all clinical events with convulsive movements (fits), blackouts (faints) or strange behaviour or feelings (turns) are epileptic seizures. A blackout is a sudden momentary loss of vision, consciousness or memory. In the absence of focal neurological symptoms or signs a cardiac cause would be the most common cause. A good history obtained from a reliable eyewitness is the cornerstone to the diagnosis of blackouts, and in the case of epilepsy and most neurological causes of blackouts, it is the only diagnostic tool required.

Vasovagal attacks occur in the presence of a definite precipitating factor such as hot weather, emotional stress and crowded areas. Various epilepsy-like clinical phenomena (e.g. tonic spasms, myoclonic jerks and automatisms, tongue biting and urinary incontinence) may accompany vasovagal attacks and simulate epileptic seizures. Reflex syncope includes cough and micturition syncope. Blackouts that occur on rising from a recumbent position should raise the suspicion of postural hypotension, and if confirmed, a meticulous search for its cause should be carried out. Episodes of torsade de pointes may be associated with brief tonic seizures due to cerebral anoxia. When they recur frequently, it may mimic status epilepticus leading to the potentially lethal administration of intravenous phenytoin.

Absences, complex partial seizures, atonic seizures, tonic seizures and simple partial seizures that predominantly involve loss of speech or memory may present as blackouts. Basilar migraine may cause vertigo and blackouts, but the simultaneous or subsequent occurrence of headache provides the clue to the diagnosis. Patients with transient global amnesia go about their daily routines normally, but subsequently have no recollection of their actions. Narcolepsy can present with brief episodes of sudden sleep that can mimic epileptic seizures. The diagnosis of pseudoseizures or non-epileptic attack disorders is now made easier through EEG-video monitoring. The attacks are seldom stereotyped and are modified according to the relevance or importance of the audience. There is usually no or little post-ictal confusion and drowsiness.

SPEAKER ABSTRACTS

Date: 21st June 2014
Time: 0930 - 1000
Epilepsy Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

USE OF NEWER AEDs AND TREATMENT MODALITIES IN MALAYSIA

Kheng-Seang LIM
Epilepsy Council of Malaysia

Zonisamide, Lacosamide and Perampanel are recently registered in Malaysia. These are newer antiepileptic drugs (AEDs) with novel mechanism of actions. A post-marketing survey, performed by the Epilepsy Council of Malaysia, showed that the newer AEDs are used mostly for refractory focal epilepsy, in both government and private hospitals, with good tolerability and effectiveness. A brief introduction will be given on the use of other newer AEDs in Malaysia, including the recent epilepsy trials on Retigabine. Updates on epilepsy surgery in Malaysia will be presented including modified video-telemetry protocol, success in ictal SPECT, usefulness of PET, and the use of intracranial EEG monitoring in Malaysia. The feasibility of a newer palliative non-pharmacological treatment, i.e. repetitive transcranial magnetic stimulation, in Malaysia will be briefly discussed.

Date: 21st June 2014
Time: 1100 - 1130
Stroke Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

PAEDIATRIC STROKE : CHALLENGES IN DIAGNOSIS AND TREATMENT

Dr. Teh Chee Ming
Hospital Pulau Pinang

Based on large epidemiological studies, stroke occurs in 2-8 per 100,000 children / year. The incidence is highest during neonatal period. The impact of childhood stroke is remarkable. It is within the top ten causes of death for infant. Besides, up to 60-70% of children with stroke sustain persistent neurological deficits.

The diagnosis of stroke in children is often missed or delayed. The major reasons are: 1) lack of awareness among care-takers and healthcare workers, 2) diverse & variable manifestations and 3) wide differential diagnoses for childhood stroke. To diagnose stroke in children, beside clinical evidence of focal neurological deficits lasting > 24 hours, a neuro-imaging showing focal ischaemic / haemorrhagic infarct compatible with vascular territory is required. The diagnosis of childhood stroke alone is not sufficient. It is always paramount to work out the contributory mechanism and possible risk factor. The two major mechanisms involve in arterial ischaemic stroke are vaso-occlusion and hypoperfusion. The risk factors include cardiogenic, vasculopathy, head and neck pathology, systemic / metabolic disorder, thrombophilic conditions etc. Cerebral sino-venous thrombosis (CSVT) has been increasingly recognized as the cause of stroke in children. Childhood CSVT tends to be multifactorial in aetiologies.

The management of childhood stroke is equally challenging as there is no universally-accepted treatment protocol due to lack of good-quality trials to date. However, few currently-available guidelines will shed some light in the treatment. The management encompasses initial stabilization and neuro-protection, anti-thrombotic therapy (anti-coagulant or anti-platelet) to prevent early propagation or late recurrence, neuro-surgical intervention and rehabilitation. Thrombolytic therapy is still considered to be experimental in childhood stroke.

International Paediatric Stroke Study (IPSS) is a global research initiative for childhood stroke, based in Toronto, Canada. This international research collaboration will definitely enhance our understanding in childhood stroke and subsequently, overcome some of the current challenges.

SPEAKER ABSTRACTS

Date: 21st June 2014
Time: 1130 - 1200
Stroke Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

ACUTE STROKE MANAGEMENT

Dr. Siva Seeta Ramaiah
Hospital Kuala Lumpur

The chain of events favoring good functional outcome from an acute ischemic stroke begins with the recognition of stroke when it occurs. Data show that the public's knowledge of stroke warning signs remains poor. Given the narrow therapeutic windows for treatment of acute ischemic stroke, timely ED evaluation and diagnosis of ischemic stroke are paramount. Hospitals and EDs should create efficient processes and pathways to manage stroke patients in the ED and inpatient settings. This should include the ability to receive, identify, evaluate, treat, and/or refer patients with suspected stroke, as well as to obtain access to stroke expertise when necessary for diagnostic or treatment purposes.

Stroke is a primary failure of focal tissue oxygenation and energy supply. Thus, it is intuitive that systemic hypoxemia and hypotension be avoided and, if present, corrected to limit further cellular damage. Intravenous fibrinolytic therapy for acute stroke is now widely accepted since the USFDA approval in 1996, in part on the basis of the results of the 2-part NINDS rtPA Stroke Trial.
The number of options for endovascular treatment of ischemic stroke has increased substantially over the past decade to include intra-arterial fibrinolysis, mechanical clot retrieval with the Merci Retrieval System, mechanical clot aspiration with the Penumbra System and acute angioplasty and stenting. Though intra-arterial therapy achieves early recanalization, these does not translates into good clinical outcome. As with intravenous fibrinolysis, time is brain for all forms of endovascular reperfusion, and all efforts must be made to reduce time to reperfusion, because the likelihood of favorable outcome is directly linked to the time to reperfusion.

Date: 21st June 2014
Time: 1200 - 1230
Stroke Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

STROKE PREVENTION: ANTI-PLATELET THERAPY AND ITS RATIONAL USE

Prof. KS Tan
University of Malaya Medical Centre

The use of antiplatelet therapy has developed significantly over the last 15 years in neurology.This lecture will cover important background literature, recent updates and evidence based recommendations on the prevention of future stroke among survivors of ischaemic stroke or transient ischaemic attack.

SPEAKER ABSTRACTS

Date: 21st June 2014
Time: 1400 - 1430
Movement Disorders Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

UPDATE ON THE MANAGEMENT OF PARKINSON'S DISEASE

Dr. Ooi Phaik Yee
Hospital Sungai Buloh

Parkinson's disease is an alpha synucleinopathy characterized by progressive loss of nigrostriatal and extranigral dopaminergic neurons, and deposition of Lewy bodies in the brain.

The Parkinson's disease timeline generally lasts 10-20 years with initial good response to treatment. Due to the degenerative nature of the disease, patient deteriorates with time and inevitably became disabled. It is not curable and till date there is no neuroprotective agent available.

Clinically Parkinson's disease manifests by motor and non-motor symptoms with variable response to treatment.

Various pharmacological agents of different mechanisms of action are developed to treat Parkinson's disease, and the list is expanding. Treatment should be individualized and the consideration differs based on the stages of the disease.

At early stage when the disease is mild, treatment is aimed for symptomatic control, as well as prevention/delay of dyskinesia and motor complications.

At moderate to advanced stage, treatment focus is on reducing the motor complications. Invasive therapy eg surgical intervention can be considered in selected patients.

For the final stage of Parkinson's disease, palliative care should be offered, engaging multidisciplinary care to provide comfort to the patients and carers.

At all stages, treatment of non-motor symptoms should also be given emphasis to reduce morbidity, though generally they are less responsive to treatment compared to the motor symptoms.

The Movement Disorders Society Evidence Based Medicine (EBM) Review Update provides the latest evidence for pharmacological and non-pharmacological treatment of Parkinson's disease. It serves as a guide for the clinicians when managing patients in their daily practice. However these should also be integrated with the clinician's experience and judgment, patient preference, expert opinion and medical economic determinants.

SPEAKER ABSTRACTS

Date: 21st June 2014
Time: 1430 - 1500
Movement Disorders Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

APPROACH TO MOVEMENT DISORDERS IN CHILDREN

Dr. Tajul Arifin bin Tajudin
Hospital Sultan Ismail

Pediatric movement disorder is field, frequently neglected, as much as it is poorly recognised. Movement disorders in children, is a common problem encountered in any neurology clinic. They constitute a heterogeneous group of disorders with variable aetiologies and clinical presentations. Though they may share some characteristics with their adult counterpart, they indeed are a unique group of disorders. This in part, is determined by the metabolic, physiological and environmental differences of the developing brain in a child. This inevitably makes the nervous system of children more vulnerable to various insults such as birth trauma and infections. Besides that, the causes can either be inherited or acquired. As a result, a systematic and meticulous approach is warranted in dealing with any child with movement disorders. The objective of this lecture is to give an overview of the basic approach to paediatric movement disorders.

Date: 22nd June 2014
Time: 0830 - 0900
Research Frontiers in Neuroscience
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

NEUROSCIENCE RESEARCH PROGRAM @ UPM

Dr. Cheah Pike See
UPM

UPM is acknowledged as the Research University (RU) and the university put research at forefront in pursuit of academic excellence. Neuroscience research is pertinent because it helps to improve understanding of human thoughts, emotions, behaviours and most importantly, the underlying mechanism of neurological disorders. In 2010, the Neuroscience Cluster of the Faculty of Medicine and Health Sciences, UPM, was formed by a small group of academicians with self-driven initiative and with different research interests in neuroscience. The cluster members are highly committed to their mission in imparting better knowledge of neuroscience to students and academics during the Monthly Neuroscience Seminar Series, annual Neuroscience Symposium, NeuroFair event and etc. The cluster members have been integrating and consolidating both clinical and lab-based research to take the field of neuroscience to another level. Later, in year 2011, the Neuroscience Research was recognised as one of the priority niche areas in UPM.

In early 2014, an inaugural meeting of Neuroscience Research Program was held and chaired by Prof. Dr. Hamidon Basri. The research program encompasses multi and transdisciplinary research on diverse disciplines in the field of Neuroscience including chemistry, computer science, engineering, linguistics, mathematics, medicine, physics and psychology. This research program aims to gather neuroscientists from various faculties of UPM, to elaborate on the main objective of the establishment of the program and to set a common goal for all. The member of the Neuroscience Research Program believe that the integration of the various disciplines will enhance the understanding of brain disease process, brain health and human behavior alongside with the application of technology in diagnostics, treatment, prevention and health promotion. This will eventually be translated into better health for the population and nation.

SPEAKER ABSTRACTS

Date: 22nd June 2014
Time: 0900 - 0930
Research Frontiers in Neuroscience
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

A PUSHY PARTNERSHIP - ENDOCANNABINOID AND KETUM *(MITRAGYNA SPECIOSA)* MISUSE POTENTIAL

M Muzaimi[1], N Jayabalan[1], NI Wan Ismail[1], H Jaafar[2], SM Mansur[3], CM Muller[4]
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[3]Centre for Drug Research, Universiti Sains Malaysia
[4]Psychiatric & Psychotherapy Centre, Erlangen University, Germany

The worldwide marketing of emerging *Mitragyna speciosa* Korth (Ketum or Kratom) pose genuine concerns, primarily owing to its opioid-like anti-nociceptive property. It is now apparent that the international use of various ketum chemotype preparations has spread beyond its traditional geographical boundaries. Despite a steady growth of data on the chemical and pharmacological properties of Ketum and its main alkaloids (mitragynine and 7-hydroxymitragynine), the neural basis for its abuse liability remains elusive. Building from the known neuropsychopharmacology of Ketum, its main alkaloids and opioid-like anti-nociceptive mirroring, along with the contemporary neurobiochemical understanding of addiction with exogenous opioids and/or cannabinoids through their intricate hijacking of the endogenous central nervous system interconnections serve as the key bases to unmask the existence of ketum abuse (and/or dependence). Our research, for the first time, had established a plausible neural basis for the abuse liability of this phytochemical and its major alkaloids by demonstrating the involvement of the endocannabinoid system in the induction of tolerance and neuroplasticity. This new insight may serve as an impetus for an international consensus for a wider social policy implication for Ketum abuse and/or dependence with further insights into the neurobiology of Ketum abuse liabilities. This also offers potential alternative therapeutic target for opioid-cannabinoid unabated misuse conundrum.

Date: 22nd June 2014
Time: 0930 - 1000
Research Frontiers in Neuroscience
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

FROM MAPPING TINY NEURONS TO MAPPING COGNITIVE PROCESSES

Dr. Azlina Ahmad Annuar
University of Malaya

UM has many groups working on diverse aspects of fundamental neuroscience. Spread across the campus, these groups explore topics such as where do certain motor neurons map to, what are the properties of particular spinal interneurons, what genes are involved in neural tube development and how synapses are formed. There are many groups also actively working on discovering new agents to promote spinal cord regeneration and preventing neurodegeneration, and on addiction using behavioural animal models. In this talk, I will highlight some of this research, in particular those representing breakthroughs in the field and are at the cutting edge of the latest technologies.

Date: 22nd June 2014
Time: 1100 - 1130
Neuromuscular Diseases Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

UPDATE ON MUSCLE DISORDERS

Prof. KJ Goh
Division of Neurology, Department of Medicine, University of Malaya, Kuala Lumpur

Muscle disorders are heterogeneous and can broadly be considered whether they are genetic or acquired myopathies. The latter may be responsive to therapy. Using case vignettes, a number of potentially treatable myopathies, both genetic and acquired, seen in the Malaysian population will be presented.

It is important to try to make as accurate a diagnosis of muscle disorders as possible, in order not to miss treatable conditions.

Date: 22nd June 2014
Time: 1130 - 1200
Neuromuscular Diseases Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

THE ROLE OF IMMUNOTHERAPY IN NEUROMUSCULAR DISORDERS

Dr. Santhi Datuk Puvanarajah
Hospital Kuala Lumpur

The utilization of immunotherapy in neuromuscular diseases remains the mainstay of treatment in the majority of inflammatory myopathies and neuropathies, as well as the neuromuscular junctional disorders. The key to successful management in this heterogenous group of diseases is the choice of effective immunotherapy at the appropriate dose, given in a timely manner. Classical immunotherapy is the most widely-used, beneficial, first-line treatment option. Selective immunotherapies, on the other hand, target specific mechanisms intrinsic to the immune system. This talk aims to address the options available to clinicians and practical applications in day-to-day clinical practice.

SPEAKER ABSTRACTS

Date: 22nd June 2014
Time: 1200 - 1230
Neuromuscular Diseases Symposium
Venue: Manhattan I, Level 14, Berjaya Times Square Hotel

EVALUATION OF A FLOPPY CHILD: FROM THE BEDSIDE TO WORKBENCH

Dr. Alex Khoo Peng Chuan
Paediatric Neurologist
Hospital Raja Permaisuri Ipoh

The approach to floppy infants is always challenging as often time is the limiting factor and investigations take time to process and parents are anxious while paediatricians are under pressure. These children often have respiratory distress and may deteriorate anytime very rapidly from birth till late infancy with significant mortality and morbidity rates. Decision making skills often come by way of clinical experience rather than factual acquisition of knowledge. In this talk , the structure as well as organised manner in which a diagnosis can be made would be emphasised. From the bedside to workbench is an apt description of the process in which clinical skills and observations may direct further tests and direction as far as therapeutics goes.

POSTER ABSTRACTS

POSTER ABSTRACTS

Abstract ID	Author	Paper Title	Page No
026	Nilesh Kumar Mitra	Evaluation of Histological and Locomotor Changes in a Mouse Model of Experimental Autoimmune Demyelinating Disorder	30
027	Tan Suk Fei	Development of Nanoemulsion Loaded with Valproic Acid with Response Surface Methodology to Penetrate The Blood-Brain Barrier	30
028	Thaarani Arumugam	Myasthenia Gravis in a Malaysian Population	30
029	Dahiru Sani	Neuroprotective Effect of Andrographolide Against Dopaminergic Neuro-degeneration Induced by Lipopolysaccharide	30

POSTER ABSTRACTS

ID 001

RAPIDLY PROGRESSIVE VASCULITIC NEUROPATHY: DIAGNOSIS AND MANAGEMENT DILEMMA

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[1]Department of Medicine, Universiti Kebangsaan Malaysia Medical Centre, Kuala Lumpur, Malaysia

Background
Peripheral neuropathy symptoms as an initial presentation of systemic vasculitis are rare. Vasculitic neuropathy usually not associated with life threatening manifestation of other systemic vasculitis. It usually rendered a slow progression and manifestation of systemic symptoms ranges from five to 11 years [1]. Precise history and clinical suspicion are essential not to miss an ANCA associated vasculitis. The dilemma in treating both ANCA associated vasculitis with concomitant infection needs a judicious decision in order to benefit the patient.

Case Presentation
We report a patient who had previous history of treated pulmonary tuberculosis, presented with one month history of bilateral foot drop. He had rapid progression of respiratory symptoms and renal impairment within one month onset of neuropathy symptom. Further investigations suggest reactivation of pulmonary tuberculosis with superimposed pulmonary aspergilosis. Other causes of neuropathy were excluded after extensive neuropathy investigations; hence ANCA associated vasculitic neuropathy with progression to systemic vasculitic diagnosis was made based on positive result of c-ANCA immunoflorecence test and vasculitic finding in the sural nerve biopsy.

Conclusion
This patient had rapid progression of vasculitic neuropathy evolved into systemic manifestation. We discuss some of the difficulties involved in the diagnosis of common presentation of bilateral foot drop and unusual presentation of rapid progression vasculitic neuropathy as well as issues of treating systemic vasculitis with immunosuppression in concomitant systemic infection.

References
1. Ravi Suppiah, Robert D.M. Hadden, Rajbir Batra et al. Peripheral neuropathy in ANCA-associated vasculitis: outcomes from the European Vasculitis Study Group trials. Rheumatology 2011, DOI:10.1093/rheumatology/ker 266.

ID 002

STROKE SCENARIO IN MALAYSIA: REPORT FROM NATIONAL STROKE REGISTRY

[1]Norsima Nazifah S, [1]Zariah AA, [2]Looi I
[1]Hospital Sultanah Nur Zahirah, [2]Hospital Seberang Jaya

Introduction
Stroke is the third leading cause of death in Malaysia and among the top contributing cause of disability. National Stroke Registry with aim to collect the data of all stroke patients admitted to Malaysia hospital and to evaluate the quality of stroke management was started since July 2009.

Method
Data of acute stroke patient who admitted to participating hospital were collected by using case report form which was then transferred to highly confidential web application. Data was analyzed by using SPSS 18. For analysis purpose, SDP were divided to four group, East Coast, North Coast, Central region, South coast and East Malaysia. The gender, age, and risk factors of patients between all groups were compared by using Chi squared test with p value less than 0.05 was considered significant. We also review the stroke subtype and outcome of patient based on stroke subtypes.

Results
Until December 2013, 6261 patients registered with median age of patient was lowest in south region group (58 year old) and significant different proportion of gender patients among region. East coast patient found to have the highest numbers of hypertension, hyperlipidemia and smoker where as diabetes was found to be the highest in north coast group. 78.3% of patients were ischemic stroke but mortality rate was seen higher in hemorrhagic stroke.

Conclusion
There were significant different with regards to age, gender and risk factors of stroke patients at different regions in Malaysia. Ischemic stroke contributed the highest cases but with lower mortality rate.

ID 003

DOES SHORT-TERM OMEGA-3 FATTY ACIDS SUPPLEMENTATION PROMOTES BENEFICIAL EFFECTS ON MEMORY AND ANXIETY LEVELS?

Hafandi Ahmad, Arzadtul Bakis Zolkaply, Nur Asmiza Mustaffa Kamal, Mokrish Ajat, Tengku Rinalfi Putra Azizan, Mohd Hezmee Mohd Noor and Wan Mastura Shaik Mohamed Mossadeq
Department of Veterinary Preclinical Science, Faculty of Veterinary Medicine, Universiti Putra Malaysia, 43400 UPM Serdang Selangor MALAYSIA

Dietary omega-3 fatty acid has been known to be associated to the improvement of brain function. The improvement of brain function depends on the type of diet and duration of supplementation. The time-length supplementation is important for metabolic actions in the body. It is a known fact that omega-3 fatty acid influences brain function in animals but little is known about the short-term effects. Therefore, this study was performed to assess the effects of short-term omega-3 fatty acid supplementation on memory and anxiety levels in mice. Seven weeks old male BALB/c mice were fed either normal pellet only (Control), normal pellet containing 10% butter (BT), normal pellet added with 6.66% menhaden oil (rich in omega-3) and 3.34% soybean oil (M3), and normal pellet added with 3.34% menhaden oil and 6.66% soybean oil (S6). After three weeks of treatment diets, mice were tested for memory function by Y-maze test and anxiety levels by elevated plus maze test. Blood plasma was collected for fatty acid analysis. Results showed that mice fed with higher omega-3 fatty acid for three weeks increased memory performance with a significantly higher total arms entries and time spent in novel arm. In addition, mice fed with higher omega-3 fatty acid for three weeks had significantly less anxiety levels with more time spent exploring the open arms in comparison to closed arms. The ratio of ω-3:ω-6 fatty acids in plasma was greater in the M3 group (10.8±2.1) than the other groups. Thus, changes in the plasma fatty acid profile after three weeks effects metabolic changes on memory and anxiety levels in mice. We conclude that time dependent must be taken into account in the use of dietary supplementation in the memory performance and anxiety levels studies.

ID 004

REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION ON REFRACTORY FOCAL EPILEPSY IN MALAYSIA, A PILOT STUDY

Joyce Siew Yong Low[1], Kheng Seang Lim[1]*, Hui Ting Goh[2], Monica Chen Mun Wo[1], Sherrini Bazir Ahmad[1], Chong Tin Tan[1]
[1] Division of Neurology, Department of Medicine, Faculty of Medicine, University of Malaya
[2] Department of Rehabilitation Medicine, Faculty of Medicine, University of Malaya

Purpose
This study was designed to evaluate the therapeutic effect of low-frequency repetitive transcranial magnetic stimulation (rTMS) on patients with refractory partial epilepsy.

Methods
Patients with refractory focal epilepsy were recruited for a 2-week high intensity (90% resting motor threshold) and low frequency (1Hz) rTMS treatment. Seizure frequency and number of interictal epileptiform discharges (IEDs) in 30-minute EEG were compared between the baseline and follow-up periods.

Results
Five patients with refractory focal epilepsy were recruited for rTMS, of which three completed the study. Seizures frequency decreased as compared with baseline level in 2/3 patients, with a mean reduction from 4.33±2.55 to 3.81±2.14. The IEDs pre and post rTMS is reduced in 1/3 patients (Patient 3), increased in patient 1 and showed no change in Patient 2. Patient 3 had an improvement in seizure frequency and scales of Symptom Checklist-90 (SCL-90), QOLIE-31 and Beck Depression inventory (BDI). Seizure frequency improved for Patient 2, but no improvement noted in the SCL-90, QOLIE-31 and BDI. Unfortunately, seizure frequency did not improve for Patient 1 but the SCL-90 and BDI score did improve.

Significance
Low-frequency high intensity rTMS could be an effective and feasible treatment option for patients with refractory focal epilepsy in Malaysia.

POSTER ABSTRACTS

ID 005

NATIONAL STROKE REGISTRY : HOSPITAL RAJA PEREMPUAN ZAINAB II KOTA BHARU, KELANTAN EXPERIENCE

Rose Izura AH, Monniaty M, Norsima Nazifah S, Ahmad Hadyan Husainy H

According to the Malaysian National Burden of Disease study conducted in 2013, stroke was the top 2 leading cause of death . In our country, stroke remained a burden financially with great morbidity loss. Malaysian National Stroke Registry (NSR) which was established in 2009 aimed to describe stroke statistics and quality care in Malaysia and to implement quality indicators for all states in this country.

Our main objectives for this study is to focus on the data of acute stroke registry conducted by our most recently joined Hospital Raja Perempuan Zainab II , Kota Bharu, Kelantan in order to help develop and improve current models for stroke prediction and management in Kelantan. This is an ongoing, prospective observational study of all the stroke cases seen at medical department HRPZII with age more than 12 years old and had onset of stroke within 2 weeks prior to admission. The data collection started from November 2011 until December 2013 with a total of 441 patients were registered with mean age of 65.3 ± 11.8 years and 55.5% were males. The racial breakdown consisted of 406 (92%) Malays, 33 (7.5%) Chinese while 12(2%) of other races. Majority of the stroke types was ischaemic (77%) with only 18.5% of hemorrhagic and TIA (3%). According to OCSP classification, 35.6% were PACI, TACI (19%), LACI (17%) and 3.4% of POCI. Most of the causes for our ischaemic stroke (TOAST classification), comprised mainly of large vessels(40%), lacunar (28%) , and cardioembolic (2.9%) . Hypertension and Diabetes were the major risk factors (75% and 37%) with 37.6% were smokers and 9.5% has hyperlipidaemia. Among all stroke patients, 7.5% has moderate disability (MRS=5), 22% moderate severe disability (MRS=4) while 12.6% has severe(MRS=6) based on Modified Rankin Scale (MRS). There were 54 (12%) death during study period and the three main causes of death were massive intracranial hemorrhage (46%), massive ischaemic stroke(26%) and sepsis secondary to stoke associated pneumonia (22%).

In conclusion, based on these findings, there was an important opportunity suggested for targeted quality improvement efforts for our in hospital stroke patients and many more need to be done.

ID 006

RECURRENT HERPES ZOSTER MYELITIS WITH POSITIVE AQP4 ANTIBODY: A CASE REPORT

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Introduction
Neuromyelitis optica(NMO) is an inflammatory autoimmune demyelinating disease of the central nervous system associated with aquaporin 4 antibodies(anti-AQP4). Various infections, bacterial and viral have been associated with this entity. Here we report a case of recurrent transverse myelitis mediated by AQP4 antibodies, preceded by varicella zoster infection.

Clinical Study
12 years old girl,

Presentation	CSF Analysis	Imaging: MRI T2	Diagnosis	Treatment
(8/10/13) Quadriparesis, Hypotonia with vesicular rash of left upper limb and pain	Protein: 0.6g/dL Oligoclonal bands: +Ve Herpes /Varicella virus PCR :- -	Long segment cervical myelitis extending to medulla	Herpes zoster myelitis	Steroids , acylovir
(15/3/14) Quadriparesis, Hypotonia, hyperreflexia with similar rash and pain	Protein: 0.64g/dL Oligoclonal bands: -Ve Herpes / Varicella PCR : -Ve	Long segment myelitis involving medulla	Recurrent herpez zoster myelitis	Steroids , acylovir

She tested positive for anti AQP4 antibody and was diagnosed with severe and recurrent long segment myelitis .This is part of NMO spectrum disorders.

Discussion
This is a rare case of recurrent myelitis mediated by anti-AQP4 without optic neuritis, preceded by herpes zoster. In view of nature of NMO, we feel any child presenting with long segment myelitis , should always be tested for anti-AQP4.

ID 007

EPILEPSY SURGERY IN UMMC

Sherrini BAZIR AHMAD[1], Noraini ISMAIL[1], Kheng Seang LIM[1], Choong Yi FONG[2], Vairavan NARAYANAN[3], Norlisah RAMLI[4], Kartini RAHMAT[4] , Chong Tin TAN[1]
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Out of 1727 patients in UMMC epilepsy database, there are 26% with refractory focal epilepsy. 45 patients (mean age 35 years old; range 3-59 years old; 62.2% male) had undergone video-EEG monitoring (VEM) from 2011-2013, of which 62.2 % were having temporal lobe epilepsy (TLE). Of those with TLE, 89.2% had a lesion in the MRI; whereas of those with extra-temporal lobe epilepsy, 81.25% were lesion-positive. 73.3% had seizures recorded during the monitoring. Ictal HMPAO-SPECT was performed in 8 patients (30.7% of all SPECTs performed). Lateralisation and localization of the epileptogenic zone were determined based on 5 modalities i.e. seizure semiology, interictal EEG abnormalities, ictal EEG onset, MRI brain lesion, and ictal SPECT hypermetabolism. One subject had presurgical intracranial subdural grid implantation and one patient had intraoperative electrocorticography (ECoG) monitoring. Concordance in 3 modalities or more was present in 38% of the patients undergone VEM. 15 patients had epilepsy surgery (73.3% male), including 4 (26.7%) who had anterior temporal lobectomy, 2 (13.3%) who had selective amygdalohippocampectomy, and 9 (60%) who had lesionectomy. 66.7% (10/14 patients) achieved good surgical outcome (Engel class 1) at 6-month follow-up.

ID 008

EMPLOYABILITY AMONG PEOPLE WITH EPILEPSY IN ASIA: A SYSTEMATIC REVIEW

M.C.M. Wo[1], K.S. Lim[1], W.Y. Choo[2], C.T. Tan[1]
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[2]*Department of Social & Preventive Medicine, Faculty of Medicine, University of Malaya, Malaysia*

Prevalence of epilepsy in developing countries has been reported to be higher than developed countries, especially in rural areas. Limited evidence suggests that prevalence of epilepsy appears to be higher in some parts of Asia, where strong social stigma presence in many Asian societies. People with uncontrolled seizure were commonly perceivedas incapable and having more difficulties in obtaining and sustaining a job. To examine the extent to which epilepsy affects employability of those in such condition, a systematic review was conducted. All published literatures from 1958 to January of 2013 were systematically searched. All abstracts were independently assessed by the two reviewers to determine eligibility of inclusion criteria. Of 95 papers reported on employment status of people with epilepsy, adjusted employed rate was ranged from 14% to 89% with mean adjusted employment rate of 58%. No significant differences in mean of employment rate by continents or among Asian countries ($p>0.05$). Contrary to belief that people having uncontrolled seizures encounter more employment difficulties, similar employment rate of 58% was found between those with uncontrolled seizure and those with controlled seizures. Results showed that person with uncontrolled seizure could be sucessful in various career development. Psycological and social factors including employers' attitude, family attitude, stigma and disclosure were dominant predictors in employability of people with epilepsy. The positive and negative factors in determine successful career in epileptic person were understudied and yet needed to be explored. Absence of universal agreement on definition of employment made comparison and interpretation of studies difficult.

POSTER ABSTRACTS

ID 009

PERIPHERAL NERVE DISORDERS IN RHEUMATOID ARTHRITIS AND ITS ASSOCIATION WITH RADIOGRAPHIC JOINT DAMAGE

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Introduction

Rheumatoid Arthritis (RA) is a chronic systemic autoimmune disease that is characterized by multiple joint inflammation that leads to progressive joint deformity and multiple extra-articular involvement. Peripheral nerve disorders are one of the extra-articular manifestations that contribute significantly to the functional limitation in patients with RA.

Objectives

To determine the association between the presence of peripheral nerve disorders with radiographic joint damage, measured using modified sharp score (MSS) in patients with RA and to compare the socio-demographic and RA disease characteristics.

Method

This was a cross sectional study involving 31 RA patients who were recruited from Rheumatology Clinic UKMMC. Presence of peripheral nerve disorders were determined by nerve conduction tests. All Patients had standardized clinical interviews to assess the presence of neuropathic and information on the socio-demographic and disease characteristics of RA were obtained from the medical records. Modified sharp score (MSS) for joint damage were calculated from the hands and feet radiographs.

Results

The frequency of peripheral nerve disorders was 22.58% (7 out of 31 patients, with entrapment neuropathy (carpal tunnel syndrome) being the most common (n=5, 71.42%), followed by sensory neuronopathy (n=1, 14.29%) and S1 radiculopathy (n=1, 14.29%). There was significant association between the presence of peripheral neuropathy with higher MSS erosion score, higher age and lower education status.

Conclusions

Despite low frequency of peripheral nerve disorders. Presence of peripheral nerve disorders were significantly associated with higher MSS erosion score, higher age and lower education status.

ID 010

ANALYSIS OF DRDS AND GRIN2B GENES POLYMORPHISMS AND THEIR ASSOCIATION WITH THE DEVELOPMENT OF IMPULSE CONTROL DISORDER AMONG MALAYSIAN PARKINSON'S DISEASE PATIENTs

Z. A. Shahidee[1,2], J. Ameerah[1,3], S. -C. Chan[4], A. X. Lee[1], M. H. Noor[1], S. Y. Lim[5], A. A. Azlina[6], P. -S. Cheah[1,7], K. -H. Ling[1,2] and M. I. Norlinah[8]

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Impulse control disorder (ICD) and behaviours (ICB) is a group of behaviour disorders which are increasingly recognised in Parkinson's disease (PD) patients, resulting from the use of dopaminergic medications, particularly dopamine agonists and levodopa. It is suggested that these medications lead to the development of ICD through abnormal modulation of dopaminergic transmission and signalling in mesocorticolimbic dopaminergic system. Various studies have reported the association of dopamine receptor (DRD) and N-methyl-D-aspartate 2B (GRIN2B) genes polymorphisms with the development of ICD in PD (PD-ICD). In this study, we employed high resolution melt (HRM) analysis to genotype eleven polymorphisms in all the five DRD genes [DRD1 (rs4532, rs4867798 and -800 T/C), DRD2 (ANKK1 rs1800497, rs104894220 and rs144999500), DRD3 (rs3732783 and rs4867), DRD4 (rs1800443) and DRD5 (rs144132215)] one polymorphism in GRIN2B (rs7301328) gene between PD patients with (cases, n = 54) and without ICD (controls, n = 43). Cases were obtained from two tertiary movement disorder centers (UKMMC, n=49 and UMMC, n=45). In both centers, the diagnosis of ICD/ ICB was made using the QUIP questionnaire. Controls were obtained from PD patients attending UKMMC who screened negative for ICB using the QUIP questionnaire. The HRM analysis showed that seven out of eleven polymorphisms [DRD1 (rs4532, rs4867798 and -800 T/C), DRD2 (ANKK1 rs1800497), DRD3 (rs3732783 and rs6280) and GRIN2B (rs7301328)] exhibited clear distinction between wild-type and variant alleles. Variants of DRD1 rs4532 (OR = 0.15; 95% CI: 0.05-0.47, p = 0.0002) was found to be associated with lower risk of developing ICD among PD patients. However, other variants in DRD1 rs4867798 (OR = 5.93; 95% CI: 1.24-28.38, p = 0.0097), DRD2/ANKK1 rs1800497 (OR = 7.09; 95% CI: 1.88-26.77, p = 0.0075) and GRIN2B rs7301328 (OR = 4.33; 95% CI: 1.49-12.59, p = 0.0043) were found to be associated with the increased risk of developing ICD among PD patients. Our findings suggest that polymorphisms of dopaminergic [DRD1 (rs4532 and rs4867798) and DRD2/ANKK1 rs1800497] and N-methyl-D-aspartate 2B (GRIN2B rs7301328) play a relevant role in the development of ICD among PD patients given levodopa and dopamine agonists.

Keywords

High resolution melts analysis, DNA polymorphism, N-methyl-D-aspartate 2B, Dopamine receptor

POSTER ABSTRACTS

ID 011

A RARE CASE OF SPORADIC CREUTZFELDT-JAKOB DISEASE WITH MRI CHANGES

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Creutzfeldt-Jakob disease (CJD), caused by the pathological accumulation of prion proteins mainly affecting cerebral cortex, is a rapidly progressing neurodegenerative disorder. Because of its notorious fatality, its diagnosis is essential in patients with rapidly progressive dementia (RPD). Here we report a rare case of sporadic CJD in a previously healthy 61-year-old Malay man. He was admitted for aspiration pneumonia secondary to recurrent episodes of choking and difficulty in swallowing. On further history, he had a 2-month history of progressive forgetfulness and altered behaviour. He had not only fluctuating conscious level but also occasional aggressive behaviour. On clinical examination, he had pseudo-bulbar palsy and myoclonic jerks were present predominantly in upper limbs. However, electroencephalography (EEG) only revealed non-specific slowing pattern. Instead, magnetic resonance imaging (MRI) brain T2 FLAIR images showed gyral hyper-intensities in the right parieto-occipital and left parietal regions. Further diffusion-weighted MRI revealed restriction on diffusion as cortical ribboning. The 14-3-3 proteins status was unknown since patient was unwilling to give consent for lumbar puncture procedure. Other investigations had excluded other reversible and non-prion causes of RPD. Hence, he was diagnosed with sporadic CJD based on the clinical history and suggestive MRI findings. Supportive treatment with antibiotics, oral risperidone and nasogastric tube feeding was commenced. Thus, our case highlights the necessity of considering CJD in the differential diagnosis of RPD and the important role of MRI to diagnose CJD in our local clinical settings.

ID 012

THE VEIN OF GALEN THROMBOSIS: AN UNCOMMON SITE OF CENTRAL VENOUS THROMBOSIS

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Introduction
Two of the most common sites of central venous thrombosis are superior sagittal sinus and transverse sinus. Thrombosis of deep cerebral venous system (DCVT) is extremely rare, particularly with the involvement of the vein of Galen. Here, we describe a case of brain magnetic resonance imaging (MRI) confirmed (DCVT) of the vein of Galen.

Case Report
A 52-year-old lady with background history of hypertension and uterine fibroid, presented with a three-day history of headache and confusion. On examination, she was confused, not orientated to time, place and person. She was afebrile and hemodynamically stable. Cranial nerve examination and fundoscopy were unrevealing. Systemic physical examination and neurological examination were normal. Routine blood investigations were unremarkable and thrombophilia screenings were negative. However, brain magnetic resonance imaging (MRI) revealed deep cerebral venous thrombosis, with hyper-intense areas seen in the head of the caudate nucleus bilaterally, the thalamus and lentiform nucleus. There was also evidence of infarct at the left temporal region. The patient was initially treated with subcutaneous enoxaparin injection 60 mg given twice daily and then converted to dabigatran 150mg twice daily. Her headache and confusion resolved and she was discharged well.

Conclusion
Although DCVT is rare, an early diagnosis remains the cornerstone towards optimal results in terms of morbidity and mortality in this potential lethal condition.

ID 013

Ts1Cje MOUSE MODEL FOR DOWN SYNDROME EXHIBITS MOTOR FUNCTION DEFICIT; AN IMPLICATION OF THE PERIPHERAL NERVOUS SYSTEM DISORDER

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Down syndrome (DS) is a chromosomal abnormality caused by presence of extra-copy of human chromosome 21. Motor dysfunction due to hypotonia is commonly seen in DS individuals and its etiology is yet unknown. In this study, we employed the Ts1Cje, a mouse model for DS to investigate the motor performance in vivo and to elucidate the role of peripheral nervous system in causing hypotonia. Forelimbs strength of two groups of mice, wild type (WT) and Ts1Cje (DS), aged P60-70 was accessed using the automated grip meter and hanging wire tests. The forelimbs strength (automated grip test) result revealed a significant difference ($P < 0.05$) between the WT (88.0 $\pm$3.08s) and DS (73.81$\pm$1.52s) group. The ability of the mice to survive the hanging position was compared and the WT survival time showed a significant difference ($P < 0.001$) from the DS. Similarly the relative number of falls was evaluated, the WT (7.892 ± 0.834) and DS (4.421 ± 0.561) and the result shows a significant difference between the groups. Thus, result of both tests showed that, the WT have a strong muscle strength than the DS group. For motor coordination, there was a significant difference ($P < 0.05$) between the WT (76.49$\pm$2.93s) and DS (68.44$\pm$3.62s) group. This result agrees with the previously reported studies performed on other mouse models of DS. This study shows that the Ts1Cje mice have weaker muscle strength and poorer motor coordination as compared with the WT mice.

ID 014

SPATIOTEMPORAL EXPRESSION PROFILING AND MOLECULAR CHARACTERISATION OF miR-344b IN THE DEVELOPING MOUSE BRAIN

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MicroRNAs are small non-coding RNAs of about 22 nucleotides that regulate gene expression through inhibition or repression processes during post-transcriptional or translational stages. Studies have shown that miRNAs play a crucial role in spatiotemporal regulation of the brain development. A recent study suggested that miR-344 family is globally expressed in a developing mouse brain. In this study, we focused to characterise the expression of miR-344b during the development of mouse brain. In situ hybridisation revealed that miR-344b is strongly expressed in the germinal layer during the early stages of mouse brain development. Its expression was more restricted in the late stage of the developing mouse brain. Later, miR-344b was not expressed in the early postnatal and mature adult brains. We then further profiled the expression level of these miRNAs in the brain and other multiple organs via RT-qPCR. Four bioinformatics tools were employed to predict the downstream target genes of miR-344b. Only genes known to be identified by 3 independent bioinformatics tools and also associated with transcription regulation and nervous system development were screened further. We found that the gene that fulfilled these criteria and targeted by miR-344b was Olig2. The targeted genes will be validated via Luciferase assay. In conclusion, miR-344b is expressed in the embryonic brain and it may play a crucial role during brain development and function.

POSTER ABSTRACTS

ID 015

EXPRESSION PATTERNS OF JAK-STAT SIGNALLING PATHWAY IN THE DEVELOPING BRAIN OF Ts1Cje MOUSE MODEL FOR DOWN SYNDROME

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Neurogenesis impairment and reduced number of neuron was observed in the human Down syndrome (DS) brain and mouse model for DS. In contrast, the number of astrocyte was increased, suggesting that dysregulation of central pathway that drive neuro-to-gliogenic switch may occur in the DS brain; specifically JAK-STAT signalling pathway. JAK-STAT signalling pathway is the key regulator of gliogenesis. The tendency of neural progenitor cells in the DS brain differentiate into astrocyte may contribute to the cognitive dysfunction seen in the DS patients. Therefore, the goal of this study is to investigate the expression patterns of Jak-Stat signalling pathway in the developing brain of Ts1Cje, mouse model for DS in comparison to the disomic mice. The whole brain of Ts1Cje (n=3) and disomic mice (n=3) was collected at embryonic and postnatal time points (E10.5, E14 and P1.5). Here, we focus on Jak1, Jak2, Stat1, Stat3 and Stat6, which have been shown to express highly or stably during early brain development. Quantitative reverse transcription PCR (RT-qPCR) revealed increase expression of Stat1 and Stat6 in the Ts1Cje brains; while the expression of Jak1, Jak2 and Stat3 were not significantly different. The expression of Stat1 was found increased at E14 and P1.5 in the Ts1Cje. On the contrary, the expression of Stat6 was found increased at E10.5 and P1.5 in DS mouse model as compared to disomic mice. Further investigation on the role of Stat1 and Stat6 may provide insight into neuro-to-gliogenic switch occur in the DS brain.

ID 016

CONSTRUCT OF A NOVEL MINIGENE TOOL REPRESENTING AN EXON OF DYSTROPHIN GENE

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Introduction

Duchenne Muscular Dystrophy (DMD) is the most frequent muscle disease in children, of an X-linked recessive affecting 1 in 3500 newborn males. Majority of cases are due to the exonic deletion of the Dystrophin gene that disrupts the open reading frame, resulting in degenerative muscle wasting. Recent evidence on further skipping of adjacent exon(s) in the situation of exonic lost may restore reading frame. Restoration of the reading frame theoretically allows conversion to a milder phenotype. Limited research had demonstrated promising use of natural compounds as exon skipping inducer. Creating a novel minigene model can be used for studying the complexity of splicing mechanism, potentially translatable into identification of therapeutic targets in various related other conditions.

Objective

To construct minigenes for determining the splicing activity of an exon in dystrophin gene using natural compounds.

Materials and methods

Exonic splicing enhancer of dystrophin minigene were identified using ESEfinder 3.0 software. Those minigenes Ex-45, Ex-51, Ex-53 and artifical minigenes with specific ESE (Art-SF2/ASF, Art-Sc35, Art-SRp40, Art-SRp55 and Art-Art-empt) were contructed before subjected to the cloning process and targeted minigene were validated using sequencing prior to transfection into HEK-293 cell line for splicing assay. The assay was again validated using 2 methods which are luciferase assay by the fluorescent signal and another method by the present of targeted size band after RT-PCR and were then confirmed by sequencing analysis.

Results and discussion

Two minigenes, Ex-45 and Ex-53, were amplified and validated using sequencing analysis. These shown correct splicing process and will be further exposed to the natural compounds. Six additional minigenes are currently being optimised as well.

Conclusion

The constructed and novel minigene had been succesfully created. The minigenes will be exposed to the some natural compounds in order to elucidate putative inhibitory actions against exonic splicing enhancers in the dystrophin minigenes.

ID 017

THE PREPARATION AND CHARACTERIZATION OF CARBAMAZEPINE-ENCAPSULATED NANOEMULSION TARGETING THE BRAIN VIA PARENTERAL ADMINISTRATION

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An attempt has been made to develop a carbamazepine-encapsulated nanoemulsion (NE) that could target human brain via parenteral administration. Oil in water (o/w) NE was prepared by using safflower seed oil and oleic acid which consist of high concentration of polyunsaturated fatty acids (PUFAs) to enhance the penetration efficiency of the NE across the blood-brain barrier, the biological barrier which impedes access of most of the neurological drugs to reach the target site of the brain. Tween-80 and lecithin were used as the surfactants while α-tocopherol was used as the scavenger for reactive oxygen species (ROS) to protect the NE from lipid peroxidation. Characterization was carried out to ensure the NE fulfilled the requirement of parenteral administration, for instance particle size, polydispersity index, zeta potential analysis, transmission electron microscopy, etc. The NE was stable for 3 months with no significant physical and chemical changes.

ID 018

THE EFFECT OF PERMANENT OCCLUSION OF COMMON CAROTID ARTERIES (POCCA) ON ELECTROENCEPHALOGRAM (EEG) IN RAT'S BRAIN

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The permanent occlusion of common carotid arteries (POCCA) causes a significant reduction of cerebral blood flow (hypoperfusion) in rats. It represents a well established experimental model in order to study neuronal damage and cognitive impairment that occurs in human dementia and Alzheimer's disease. Electroencephalogram (EEG) has been used to study the function of brain by recording brainwaves during controlled behaviour in human and laboratory animals. In the present study, we evaluated the effect of POCCA and sham-operated rats on EEG which were recorded on day 7, 14, 21 and 28. Male Sprague-Dawley rats (250-300g) common carotid arteries were permanently ligated. The electrodes were implanted on frontal, sensory, hippocampus left and right and the EEG activity was recorded in freely moving rats. The activity was recorded, amplified, filtered (band pass filters to separate and display the frequency bands: Delta, 0.1-3.5 Hz; Theta, 3.6-7.5 Hz; Alpha, 8-13 Hz; Beta, 14-30 Hz) and digitized (200Hz) by PowerLab/4s system. The POCCA rats on the frontal region shows suppression of brain activity up to day 21, but, day 28 resulted increase in theta, alpha and beta bands. The same results were found in both hippocampus. However, on sensory region, the suppression of alpha and beta bands were prominent on day 28. Large increase of theta activity on day 28 in both frontal and hippocampus regions could be linked to subcortical damage as seen in mild vascular dementia. The suppression of brain activity seen before day 28 may be reflected by the involvement of other monoaminergic systems.

POSTER ABSTRACTS

ID 019

BEHAVIOURAL EFFECTS AND MOTOR COORDINATION IN SWISS ALBINO MICE EXPOSED TO CHRONIC TREATMENT OF MORPHINE AND MITRAGYNINE (KETUM PHYTOCHEMICAL)

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Background
Worldwide, extracts from numerous natural products as phytochemicals had been subjected to a growing misuse as psychoactive substance. Phytochemicals such as that from opioids and Mitragynine (from Ketum, or Mitragyna speciosa) are recognised for their high addictive liability, along with behavioural, motivation and motor system effects.

Objective
This study aimed to evaluate the behavioural changes induced by Morphine and Mitragynine (MG).

Methods
We analysed mice behaviour in social groups using the Intellicage® learning system and the rotarod motor coordination. Eighteen male Swiss albino mice were randomly housed in groups of six. Mice were subjected to a 28-days regimen with morphine sulphate (5 mg/kg, s.c.) and MG (5 to 25 mg/kg, i.p). Control group received Tween-20 vehicle (1 ml/kg, i.p.).

Results
Differences in exploratory activities were significant where the Morphine and MG group showed an increase in the activity compared to their baseline and the control group activity. In the sucrose preference paradigm, Morphine and MG treated animals exhibited significantly higher sucrose preference level. Meanwhile, the control animals showed somewhat moderate level of likeliness for the sweetened water. The association of the sucrose reward corner with air-puff punishment, however, does not decrease the degree of sucrose seeking behaviour in the treated animals. In contrast, vehicle treated animals showed significant aversion towards sucrose. In the place and reversal learning paradigms, the psychoactive compounds treated animals failed to perform operant learning task and to acquire the water-rewarded corner and spatially-shifted corner. On the other hand, the Morphine and MG treated animals also showed difficulties in managing the accelerating rod. However, the degree of latency of fall was higher in MG treated compared to Morphine treated group. The vehicle treated mice did not show any difference compared to their baseline performance.

Discussion/Conclusion
Our results suggest chronic misuse of Morphine and MG results in lasting neuroadaptive effect resulting in hyperactivity, hedonism, impairment of learning and memory and motor coordination. Future study to deliberate the underlying neural basis for these changes is warranted, particularly in relation to the emerging Ketum misuse.

ID 020

BEHAVIOURAL AND NEURONAL ALTERATIONS IN SWISS ALBINO MICE FOLLOWING CHRONIC MITRAGYNINE AND MORPHINE TREATMENT

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Background
Mitragynine is the major alkaloid compound of Mitragyna speciosa (Ketum), a plant native to the northern region of Malaysia and southern region of Thailand. Literature indicates that Mitragynine produce effects similar to morphine, hence subjects to addictive liabilities. Behavioural and neuronal changes have been associated with chronic morphine exposure. The aim of this study is to investigate detailed behavioural and neurological adaptations in mice brain reward pathway, particularly involving cannabinoid (CB_1) receptor, produced by mitragynine and morphine sensitisation.

Methods
Male Swiss albino mice were subjected to a 28-day regimen with Tween20-vehicle (1ml/kg, ip, n=6), mitragynine (5-25mg/kg, ip, n=6) or morphine sulphate (5mg/kg, sc, n=6). IntelliCage® was used as the setting for context-independent behavioural sensitisation to observe mice exploratory behaviours, motivation and persistence for natural reward, spatial learning and reversal learning. On Day-29, mice brains were removed and processed for immunohistochemistry and Western Blot analyses for the expression of CB_1 receptor.

Results
Mitragynine and morphine sensitisation significantly increased horizontal locomotion ($p<0.005$), preference for sucrose ($p<0.005$), and persistence in sucrose-seeking despite air-puff punishment ($p<0.005$). Impairment in spatial learning and reversal learning were also observed in both groups ($p<0.05$). In immunohistochemistry and Western Blot test, chronic mitragynine and morphine resulted in the up-regulation of CB_1 receptors in hippocampal CA1 region ($p<0.05$), nucleus accumbens ($p<0.001$) and amygdala ($p<0.001$), the key brain reward region.

Conclusion
These findings demonstrate the role of brain CB_1 receptors in the behavioural and neural plasticity with chronic mitragynine and morphine use.

ID 021

Δ^9-THC PROMOTE NEUROGENESIS IN TREATED SPRAGUE DAWLEY RAT

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Cannabis, a genus of plant inclusive Cannabis indica and Cannabis sativa, has a euphoric property and commonly prepared as ganja, hashish and marijuana. Cannabidiol was reported to cause modulation of Δ^9-THC. Administration of Δ^9-THC led to neurodegeneration observed in brain by activation of CB_1 receptor. Despite that, treatment of Δ^9-THC among schizophrenic patient showed positive effects on neuroplasticity. With those controversial findings, this study was designed to determine the optimum dose required to boost the positive effects while minimize adverse effects on the neuroplasticity development. For this study, three doses of Δ^9-THC were used; 0.75, 1.5 and 3.0 mg/kg, for 21 days. 3% of ethanol with 0.9% of normal saline was used as a control. Brain was collected and fixed for immunohistochemistry (IHC) technique. Different markers represent neurogenesis process were used; BrdU, GFAP, nestin, DCX, and TuJ-1. The analysis was done using SPSS 18.0 for one-way ANOVA and Tukey's multiple comparison tests with significant < 0.05. The scoring was focused on the hippocampus region, where the adult neurogenesis was known to occur. As the result, 1.5 mg/kg of Δ^9-THC showed significant different compared to control, 0.75, and 3.0 mg/kg of Δ9-THC ($p < 0.05$) for all markers. This study had postulated the effects of Δ^9-THC on neuroplasticity in the hippocampus. Δ^9-THC was showed to promote neurogenesis through increment in proliferation, differentiation, migration, targeting, and synaptic integration of neuronal cell.
Keywords: Δ^9-THC, neuroplasticity, adult neurogenesis

POSTER ABSTRACTS

ID 022

ATRIAL FIBRILLATION : A MAJOR CONTRIBUTOR TO STROKE SEVERITY AND POORER OUTCOME IN ACUTE ISCHAEMIC STROKE PATIENTS IN MALAYSIA

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Background
Atrial fibrillation (AF) is an independent risk factor for stroke and a important prognostic factor for poor outcome. There is a paucity of data on AF and stroke in Malaysia.

Objectives
To investigate the clinical profile, risk factors and clinical outcome in AF-associated acute ischaemic stroke (AF-AIS) compared to ischaemic stroke patients without AF (AIS) and to determine the prevalence of AF among ischaemic stroke patients.

Methods
This study consisted of 2 phases. A total of 200 ischaemic stroke patients were admitted to the neurology ward of the University of Malaya Medical Centre. A retrospective study was performed on the initial 100 consecutive ischemic stroke patients followed by a prospective, observational cohort study on the subsequent 100 consecutive ischemic stroke patients.

Results
The prevalence of AF-AIS was 15.4%.The AF-ASIS were older; 68.26 vs 62.25, [p<0.05, OR 2.53 (1.01-11.01)]. 61.3% of the AF-AIS were male. All major racial groups were represented. 12/31 (38.7%) of the AF-AIS had chronic AF; 2/12 (14.3%) were not on treatment, 4/12 (33.3%) demonstrated poor time in therapeutic range(TTR) and the remaining 5/12 (41.7%) defaulted treatment due to financial constraints and bleeding complications.Hemorrhagic transformation was significantly higher among AF-AIS; 38.7% vs 7.1%, [p<0.05, OR 0.12 (0.05-0.31). A higher percentage of AF-AIS had poorer modified Rankin score (3-6) on discharge and higher mortality; 80.6% vs 57.7%, [p<0.05, OR 3.05 (1.19-7.82)] and 19.4% vs 4.2%, [p<0.05, OR 5.52(1.72-17.77)] respectively.

Conclusion
AF-AIS was associated with poorer outcome and higher mortality. Strategies to improve compliance to anticoagulation therapy should be implemented.

ID 023

DIFFERENTIATION POTENTIAL OF ADULT HUMAN MESENCHYMAL STEM CELLS DERIVED FROM ADIPOSE TISSUE, TEETH PULP AND WHARTON'S JELLY INTO NEURONAL-LIKE CELLS

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As multipotent progenitors, mesenchymal stem cells (MSCs) from adult tissues have the ability to differentiate into various lineages, including neuronal cells. Employing adult MSCs for neuronal cells generation is potentially significant in regenerative medicine therapy to treat neurological disorders, such as Parkinson's disease, besides circumventing bioethical issues associated with embryo derived stem cells. However, establishment of specific assured protocols is still in its infancy; hence, this study which investigated the potential of MSCs from different adult human sources to generate phenotypes of neuronal lineages in vitro. MSCs were isolated from adipose tissue, Wharton's jelly of the umbilical cord tissue (hUCMSCs) and teeth pulp obtained with informed consent from UMMC and Dentistry clinic patients, respectively. Isolated MSCs were cultured and maintained in complete medium and incubated at 37ºC. Media was replaced every three days and the cells were subcultured on reaching 70%- 80% confluency. Passage 5 MSCs were initially treated for 24 hours with different concentrations (0.2µl, 0.3µl and 0.4µl) of β-mercaptoethanol (β-ME) to induce neuronal differentiation. After 24 hours treatment, media was changed and replaced with fresh serum free media. MSCs were then treated with higher β-ME concentrations (2.0µl, 3.0µl and 4.0µl) for 3 hours. Upon exposure, adipose and teeth pulp derived MSCs displayed neuronal-like phenotype as detected under phase contrast microscopy. However, the protocol did not work for hUCMSCs. Thus, the findings propose the current protocol as suitable to differentiate human adipose tissue and teeth pulp MSCs into neuronal-like cells.

ID 024

A REVIEW OF PLASMAPHERESIS FOR NEUROIMMUNOLOGICAL DISORDERS IN SARAWAK

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Introduction
Plasmapheresis is an established therapeutic option in many autoimmune disorders. The removal of pathogenic autoantibodies through extracorporeal filtration of plasma forms the basis of plasmapheresis technique.
OBJECTIVES: This study reviews the use of plasmapheresis in our centre.

Methodology
A total of 9 patients and 57 plasmapheresis sessions between June 2012 and May 2014 were identified. Demographics and clinical data including indication, use of combination therapy and regime were recorded. Primary outcome was clinical improvement as assessed by Clinical Global Impression (CGI) improvement score of <3 at discharge.

Results
There were 7 females and 2 males. Median age was 32 years (range 14 to 59). Indications include transverse myelitis (n=5), myasthenia gravis (n=2) and autoimmune encephalitis (n=2). Reasons for plasmapheresis include poor steroid response, severe spinal attacks and intravenous immunoglobulin (IVIG) anaphylaxis. 8 patients received additional therapy of IVIG and/or methylprednisolone. Majority received 5 exchange sessions. Only one patient with refractory myasthenia gravis complicated with IVIG allergy required repeated sessions. 1 to 1.5 times plasma volumes were exchanged per session every other day, and substituted with 5% albumin Hartmann. Systemic heparin was used for anticoagulation. Clinical improvement (CGI 1-3) was achieved in 78% (n=7) while 22% (n=2) showed no improvement (CGI 4). None deteriorated post plasmapheresis.

Conclusions
Our data showed that plasmapheresis has a promising role in various neuroimmunological disorders either as first-line or adjunctive therapy.

ID 025

INTRAVENOUS IMMUNOGLOBULIN USE IN NEUROLOGY UNIT, SARAWAK GENERAL HOSPITAL

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Introduction
Intravenous immunoglobulin (IVIG) has the potential to modulate numerous different effectors of autoimmune diseases via multiple action mechanisms. This has led to escalation of use in various off-label indications although the Food and Drug Administration (FDA) approved IVIG use in chronic inflammatory demyelinating polyneuropathy (CIDP) and multifocal motor neuropathy only.

Objectives
The objectives of this study are to analyse the pattern of IVIG use across indications and the proportion of appropriate off-label indications.

Methodology
A total of 50 neurology patients receiving IVIG between September 2011 and May 2014 were identified. Demographic data, indications and dosage regimen were analyzed retrospectively. Subanalysis on off-label indications were based on American Association of Neurology (AAN) 2012 guideline.

Results
Male-to-female ratio was 1:1. Median age was 44 years (range 15 to 76). IVIG was prescribed for Guillain-Barre syndrome (42%), myasthenia gravis (30%), CIDP (12%), autoimmune encephalitis (8%), other neuropathy (4%), paraneoplastic disorder (2%) and polymyositis (2%). Majority received IVIG 0.4g/kg/day for 5 days as a single course. Only 5 patients received repeated IVIG treatment (between 2 to 8 courses). Off-label indications constitute the mass of IVIG use. The overall IVIG use however, was deemed appropriate by AAN guideline definition (84%,n=42).

Conclusions
The potential use of IVIG in a wide range of neurological conditions is expanding. Given the costs and its implication on health economics, a careful review on available evidence and local guidelines are warranted.

POSTER ABSTRACTS

ID 026

EVALUATION OF HISTOLOGICAL AND LOCOMOTOR CHANGES IN A MOUSE MODEL OF EXPERIMENTAL AUTOIMMUNE DEMYELINATING DISORDER

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Introduction
Multiple sclerosis (MS) is an autoimmune disorder characterised by demyelination of the central nervous system. The aetiopathology of MS is still not clear. The objective of this study was to produce an animal model of MS, in the form of experimental autoimmune encephalomyelitis (EAE).

Methods
Five C57BL/6 mice (10 weeks) were immunized with 250 µl of MOG35-55, a peptide with Freund's adjuvant. Inactivated Pertussis toxin was injected via intraperitoneal route on day 0 and day 2. The control mice (n=5) were given 250 µl of PBS subcutaneously. Video recording of locomotion and footprint pattern analysis was done till 20th day of experiment. Paired sample t-test was done to find out difference between pre-experimental and post-experimental data. On the 20th day, the mice were sacrificed under anaesthesia. Paraffin sections of brain, spinal cord and sciatic nerve were stained with Luxol Fast Blue (LFB) stain and H& E stain.

Results
The immunized mice developed loss of tonicity of tail and weakness of hind limb on 2nd week. The parameters of average distance covered, velocity, right stride length and left stride length were reduced on day 6 and 14. Left stride length of days 3, 6, 14 and 20 in immunized group were negatively correlated with pre-experimental baseline data ($p<0.05$).The LFB staining showed few areas of demyelination over posterior white column of the lumbar part of spinal cord. H & E staining showed dilatation of the lateral ventricles and inflammatory cells in sections of sciatic nerve.

Conclusion
Immunization with MOG produced isolated areas of demyelination in spinal cord of mouse. The locomotion of left lower limb was found to be impaired marginally in the form negative association between post-experimental and pre-experimental left stride length.

ID 027

DEVELOPMENT OF NANOEMULSION LOADED WITH VALPROIC ACID WITH RESPONSE SURFACE METHODOLOGY TO PENETRATE THE BLOOD-BRAIN BARRIER

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Nanoemulsion was used to "piggyback" valproic acid drug to improve the pharmacokinetic profile of the drug. Response surface methodology (RSM) with central composite design was used to optimize the processing method of high energy ultrasonic such as pre-sonication ultrasonic intensity (A), irradiation time (B) and temperature (C) on the nanoemulsion loaded with valproic acid. Influence of the aforementioned ultrasonic processing parameter on the responsive parameter such as particle size, zeta potential and polydispersity index were studied. From the analysis, it is found that the interaction between ultrasonic intensity and irradiation time is the most influential factor on the particle size of nanoemulsion formulated. With this optimization method, the lowest particles size with reasonable polydispersity index in the shortest time was obtained. A valproic acid loaded nanoemulsion can be obtained with 60% power intensity for 10 minutes at 50º C. Particles size of 43.21±0.11 with polydispersity index of 0.211 and zeta potential of -21.87mV±0.35 were produced. Accelerated stability studies suggest a good stability of formulated nanoemulsion loaded with drug as there is no significant changes in physicochemical aspect such as particle size, polydispersity index and zeta potential value. Apart of that, transmission electron microscope was employed to determine the shape of the nanoemulsion. Thus with the all aforementioned characteristic optimized, the formulated nanoemulsion is believed to has the potential to penetrate blood-brain barrier in the treatment of epilepsy.

KEYWORDS: nanoemulsion, valproic acid, epilepsy, RSM, CCD, ultrasonication, blood-brain barrier

ID 028

MYASTHENIA GRAVIS IN A MALAYSIAN POPULATION

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Introduction
Whilst the epidemiology of myasthenia gravis (MG) is well-described in the Western population, there are few reports of MG in South-East Asia. In the current study, we aimed to study the pattern of MG in a large cohort of patients presenting to a tertiary neurology Centre

Methods
From an existing MG registry, we identified 196 patients with MG presenting to University Malaya Medical Centre. The demographics, disease characteristics and outcomes were reviewed and presented here.

Results and Discussion
We found females predominate at a ratio of 1.45:1, presenting at a younger age. The median age of diagnosis was within the 41-50 age group and the typical bimodal presentation was not seen. Patients of Malay ethnicity presented at a younger age whereas Chinese patients were older at diagnosis. The MG types were mainly generalized MG (58%) followed by ocular (33%) and ocular to generalized (9%). In comparison to generalized MG, patients with ocular disease were in the older age group (>40 years). Acetylcholine receptor antibody testing was performed in 165 patients and of these, 76% were seropositive. Thymectomy was performed in 76 (39%) patients and in 55 patients, thymus data were available. Thymoma was seen in 45% of patients; the majority was men and the ages ranged between 20-70 years. Thymic hyperplasia was seen in 44%, mainly females and all were under the age of 50. Very few (5%) achieved complete remission.

ID 029

NEUROPROTECTIVE EFFECT OF ANDROGRAPHOLIDE AGAINST DOPAMINERGIC NEURO-DEGENERATION INDUCED BY LIPOPOLYSACCHARIDE

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Neuroprotection is distinctly define as the 'protection of neuron" and strategically used to protect and salvage the brain from death in a number of different neuro-degenerative disorders like Parkinson's disease, traumatic brain injury, ischemic stroke. Complex cascade of events have been shown to occur, eventually leading to death of neuron. Inflammation and oxidative stress triggers the production of toxic pro-inflammatory cytokines and free radicals leading to cellular degeneration associated with many chronic and degenerative disorders. However, down-regulation of inflammatory and oxidative stress markers offers defence against development and progression of the disease. Andrographolide (AGP) is the most abundant diterpene lactone isolated from the leaves of Andrographis paniculata. Despite the increasing literature studies on the anti-inflammatory effect of AGP, there is still paucity regarding its neuroprotective role as can be ascertained from the survey of scientific literature. The present study investigated the potential therapeutic role of AGP as a neuroprotective agent via in vitro model of lipopolysaccharide (LPS)-induced brain injury using microglial (BV2) and dopaminergic (N27) cells. Pre-treatment of AGP at non-cytotoxic concentration range (0.25-2.0 µM) of BV2 cells followed by LPS dose-dependently suppressed nitric oxide production, pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) as well as attenuating intracellular reactive oxygen species (ROS) and thiobarbituric acid reactive substance (TBARS) compared to cells without AGP pretreatment. Further, conditioned media (CM) of the pretreatment had diminished toxicity towards dopaminergic neurons as compared to CM without pretreatment.

Keywords: Andrographolide; lipopolysaccharide; oxidative brain injury; dopaminergic neuro-degeneration.

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1. Lopez OL et al. J Neurol Neurosurg Psychiatry 2009:80(6):600-607

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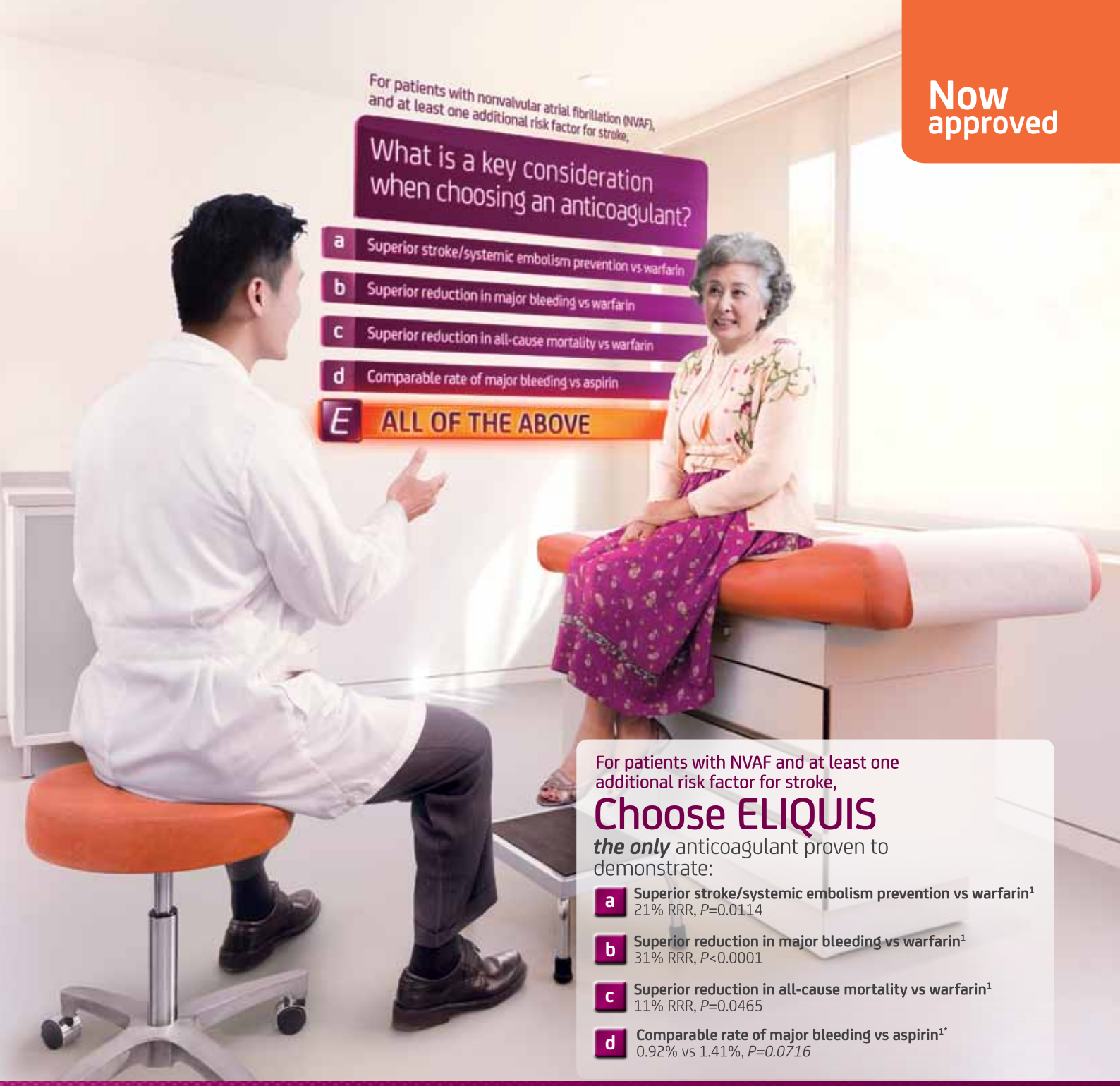
Date of revision : June 2012
Full prescribing information is available upon request.



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*Relative risk reduction.

ABBREVIATED PRESCRIBING INFORMATION

PFIZER

CONTENT: Apixaban • **INDICATION:** Prevention of stroke and systemic embolism in adult patients with non-valvular atrial fibrillation (NVAF), with one or more risk factors, such as prior stroke or transient ischaemic attack (TIA); age ≥ 75 years; hypertension; diabetes mellitus; symptomatic heart failure (NYHA Class ≥ II). Prevention of venous thromboembolic events (VTE) in adult patients who have undergone elective hip or knee replacement surgery • **RECOMMENDED DOSAGE:** Recommended 5 mg bd for prevention of stroke & systemic embolism in patients with NVAF. 2.5 mg bd for NVAF patients with at least 2 of the following characteristics: age ≥ 80 years, body weight ≤ 60 kg, or serum creatinine ≥ 1.5 mg/dl (133 micromole/l). 2.5 mg bd for Prevention of VTE. Initial dose should be taken 12-24 hr after surgery. Recommended duration of treatment: Patient undergoing hip replacement surgery 32-38 days. Patient undergoing knee replacement surgery 10-14 days. • **ADMINISTRATION:** Take with or without food • **CONTRANDICATION:** Hypersensitivity. Clinically significant active bleeding. Hepatic disease associated with coagulopathy & clinically relevant bleeding risk. Lesion or condition at significant risk of major bleeding (eg current or recent GI ulceration, presence of malignant neoplasms at high risk of bleeding; recent brain/spinal injury, brain, spinal/ophthalmologic surgery, recent intracranial haemorrhage; known or suspected oesophageal varices, arteriovenous malformations, vascular aneurysms or major intraspinal or intracerebral vascular abnormalities. Concomitant treatment with any other anticoagulant example unfractionated heparin (UHF), low molecular weight heparins, heparin derivatives, oral anticoagulants except under the circumstances of switching therapy to or from apixaban or when UHF is given at doses necessary to maintain a patent central venous or arterial catheter. • **SPECIAL PRECAUTIONS:** Increased risk of hemorrhage. Discontinue if severe hemorrhage occurs. Concomitant use with anti platelets; NSAIDs including acetylsalicylic acid; other platelet aggregation inhibitors (following surgery); strong CYP3A4 & P-gp inhibitors (eg azole antimycotics & HIV-Protease Inhibitors) & inducers. Use of thrombolytic agents for the treatment of acute ischemic stroke. Discontinue at least 48 hr prior to elective surgery or invasive procedures with a moderate or high risk of bleeding; at least 24 hr prior to elective surgery or invasive procedures with a low risk of bleeding. Temporary discontinuation. CrCl <15 mL/min or those undergoing dialysis; mild or moderate & severe hepatic impairment; elevated liver enzymes ALT/AST >2x ULN or total bilirubin ≥ 1.5 x ULN. Rare hereditary problems of galactose intolerance, Lapp lactase deficiency or glucose-galactose malabsorption. Pregnancy & lactation. Elderly (co-administration with acetylsalicylic acid). Spinal/epidural anaesthesia or puncture; hip fracture surgery. • **ADVERSE REACTIONS:** Anaemia (including postoperative and haemorrhagic anaemia, and respective laboratory parameters), Haemorrhage (including haematoma, and vaginal and urethral haemorrhage), Nausea, Epistaxis, Contusion, Haematuria, Haematoma; Eye haemorrhage (including conjunctival haemorrhage)& Gastrointestinal hemorrhage (including haematemesis and melaena), rectal haemorrhage gingival bleeding. • **DRUG INTERACTIONS:** CYP3A4 & P-gp inhibitors: Increased plasma conc with azole antimycotics (eg ketoconazole, itraconazole, voriconazole & posaconazole) & HIV-PIs (eg ritonavir); diltiazem, naproxen, amiodarone, verapamil, quinidine. CYP3A4 & P-gp inducers: Decreased plasma conc with rifampicin; phenytoin, carbamazepine, phenobarb or St. John's wort. Increased bleeding risk with other anticoagulants, platelet aggregation inhibitors & NSAIDs including acetylsalicylic acid. Serious bleeding risk with thrombolytic agents, GPIib/IIIa receptor antagonists, thienopyridines (e.g., clopidogrel), dipyridamole, dextran and sulfinpyrazone. Reduced exposure with activated charcoal. • **PRESENTATION AND PACKAGING:** Film Coated tablet 2.5 mg x 30' and 5 mg x 20's, 5 mg x 60's • **PREG SAFETY (US):** B

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